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REVIEW ARTICLE

Light Control of Seedling Morphogenetic Pattern

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INTRODUCTION

Plant development is characterized by a high degree of plasticity in response to environmental signals. As sessile organisms, plants cannot actively move away from sources of stress, nor can they seek out a location with optimal nutrient and light resources. Instead, they must tailor their developmental pattern in a way that maximizes their chances of survival and reproduction. A plant's "choice" of developmental pattern is based largely on environmental cues, one of the most important of these being light. Given the importance of photosynthesis to plant survival, it comes as no surprise that higher plants respond to light signals by assuming a growth pattern that enhances their access and exposure to light. This control of plant form by ambient light conditions is generally termed photomorphogenesis (Kendrick and Kronenberg, 1994).

The light environment in nature is complex. Unobstructed sunlight consists of a wide continuum of photon wavelengths that is conveniently divided into three large spectral domains: UV (<400 nm), visible (400 to 700 nm), and far-red (>700 nm) light (Figure 1A). The spectral quality, or relative photon distribution, at different wavelengths can vary greatly, depending on the location and the time of day. For example, within the canopy, the light available is essentially depleted in the visible and UV regions, and far-red light is highly represented (Figure 1A). Furthermore, twilight normally has a higher far-red to red ratio than daylight (Smith, 1994). Although higher plants effectively utilize only visible light for photosynthesis, they have the capability to sense and respond to a much wider range of the spectrum, including UV and far-red light. For example, the effectiveness of different wavelengths of continuous light at inhibiting hypocotyl elongation of dark-grown *Sinapis alba* seedlings (Beggs et al., 1980) is shown in Figure 1B. It is evident that multiple spectral regions of light, including blue, red, and far-red, all are very effective at inhibiting hypocotyl elongation, suggesting that *S. alba* seedlings are capable of perceiving all of these light signals and utilizing them to control seedling morphogenesis.

Plant responses to light are especially evident in the young seedling, although they occur throughout the life of the plant. Typical responses of Arabidopsis seedlings to variations in ambient light conditions are depicted schematically in Figure 2. Under unobstructed direct light, a seedling develops according to the characteristic photomorphogenic pattern (Figures 2A and 2D), that is, it has open, expanded cotyledons and a short hypocotyl. This developmental pattern rapidly establishes the seedling as a photoautotrophic organism, and most of the plant's energy is devoted to cotyledon and leaf development, while longitudinal extension growth is minimized.

Under conditions in which light quality and intensity are reduced by shading or obstruction, a seedling develops according to the somewhat different developmental pattern shown for the shade-avoiding seedling (Figures 2A and 2E). The shade-avoiding seedling displays reduced cotyledon expansion relative to the seedling grown in unobstructed light, and hypocotyl extension is markedly increased. This increase in hypocotyl extension, which correlates with the degree of shading, allows the plant to grow up through a canopy into direct sunlight. This developmental response involves an increase in hypocotyl elongation coupled with a reduction in cotyledon and leaf expansion. The shade avoidance response can also be elicited by reflected light from neighboring plants, which may give the plant an advantage in competing for limited light resources. Plants can also respond to directional light phototropically by bending and growing toward the light (Figure 2A), thereby maximizing leaf exposure to light. Finally, if a seedling grows in complete darkness, it develops according to the etiolated pattern (Figure 2A, right). The cotyledons remain closed and unexpanded, and the hypocotyl becomes extremely elongated. This developmental response, called skotomorphogenesis or etiolation, allows a buried seedling to grow up through a soil layer to reach the light as rapidly as possible. The seedling therefore devotes its limited stored energy resources almost exclusively to hypocotyl extension.

The characteristic photomorphogenic, shade-avoiding, and etiolated seedlings can be viewed as representing a continuous developmental series or gradient in response to diverse light cues. Exactly where an individual seedling falls on this developmental gradient depends on its particular light environment, that is, on both the quantity and quality of the light

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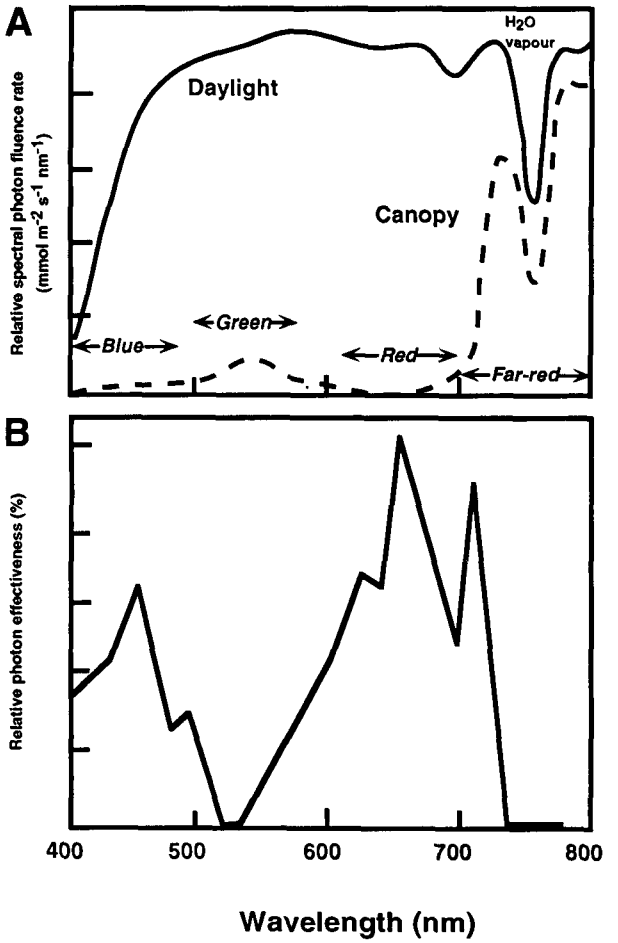

Figure 1. Spectral Photon Distribution of Sunlight and Its Effectiveness in Modulating Plant Development.

(A) Typical spectra of unobstructed daylight (solid line) and within a vegetation canopy (dotted line). The major spectral regions in the visible (i.e., blue, green, and red) and the far-red regions are indicated. Within deep canopy, blue and red light are essentially depleted, and far-red light is abundantly represented. The deep trough in the far-red portion of the daylight spectrum is caused by absorption of water vapor. Adapted from Smith (1994).

(B) The relative effectiveness of different wavelengths of continuous light for inhibition of hypocotyl elongation of dark-grown *S. alba* seedlings. *S. alba* is a close relative of Arabidopsis in the mustard family. Blue, red, and far-red light are most effective, as indicated by the three major peaks. Adapted from Beggs et al. (1980).

it receives. The less light the seedling receives, the more etiolated its morphology becomes; the more light the seedling receives, the more it comes to resemble the characteristic photomorphogenic seedling. The developmental pattern followed by a seedling is also highly flexible and adjusts in the face of changing light conditions. For example, when an etiolated seedling is exposed to light, it rapidly terminates skotomorphogenic development and initiates photomorphogenic development (Kendrick and Kronenberg, 1994). When a photomorphogenic seedling grown in unobstructed light is exposed to shade light, it displays shade-avoiding characteristics during subsequent development.

Recently, much effort has been directed toward learning about the genetic basis of light control of seedling morphogenesis. A general theme that has emerged from these studies (see Figure 3) is that a complex array of photoreceptors and possibly early signaling events are responsible for sensing environmental light cues. These signals are then integrated by the seedling to control its cellular development and morphogenetic pattern. This review summarizes recent progress in analyzing key components involved in sensing, transducing, and integrating light signals, and it attempts to correlate this information with whole-plant photomorphogenic developmental patterns and strategies. In the next section, we review the photoreceptors and immediate downstream signaling molecules that sense specific light stimuli. The third section discusses important developmental regulatory molecules that may represent converging points for early light signaling and whose activities are modulated by light signals. In the final section, some of the emerging models and possible future directions of photomorphogenesis research are discussed. For brevity, we have omitted a comprehensive discussion of light-regulated gene expression and light signal transduction; the interested reader is referred to several recent reviews of these areas (Bowler and Chua, 1994; Deng, 1994; Liscum and Hangarter, 1994; Millar et al., 1994; Quail, 1994; Whitelam and Harberd, 1994; and Quail et al., 1995). Also, we do not discuss phototropic responses, because both photoreceptors and possibly downstream regulators involved in directional growth in response to light appear to be non-overlapping with those responsible for direction-independent light-mediated development (Liscum and Hangarter, 1994; Short and Briggs, 1994; Liscum and Briggs, 1995).

LIGHT PERCEPTION AND EARLY SIGNALING

The fact that different spectral regions of light are capable of eliciting photomorphogenic seedling development (Figure 1B) led to the realization that multiple photoreceptors are responsible for detecting the different wavelengths of light. These photoreceptors include the phytochromes (Furuya, 1993; Vierstra, 1993; Quail, 1994), which absorb mainly red and far-red light, the blue light photoreceptors (Ahmad and Cashmore, 1993; Kaufman, 1993), and the UV light photoreceptors (Kendrick and Kronenberg, 1994). Stimulation of any one of these three photoreceptor classes alone or in combination can induce seedling photomorphogenic development.

Perception of Red and Far-Red Light

The phytochrome family of photoreceptors is primarily, if not solely, responsible for sensing the red and far-red regions of the spectrum. All phytochromes consist of an apoprotein and a covalently attached linear tetrapyrrole chromophore.

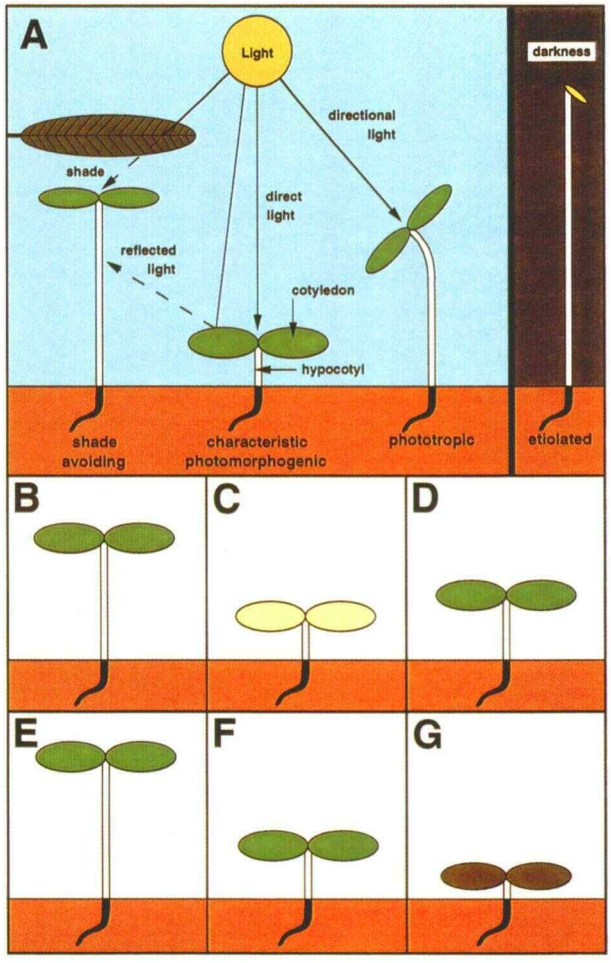

Figure 2. Schematic Diagrams of Young Wild-Type Arabidopsis Seedlings Grown under Various Light Conditions.

(A) Representative photomorphogenic developmental patterns of *Arabidopsis seedlings grown under various white light environments.*
(B) Seedling grown in continuous red light. Red light causes robust cotyledon expansion but does not inhibit hypocotyl elongation as effectively as some other wavelengths of light.
(C) Seedling grown in continuous far-red light. Far-red light at high intensities causes dramatic cotyledon expansion and strongly inhibits hypocotyl elongation. The cotyledons are white because shorter wavelength light energy is needed for the completion of chlorophyll biosynthesis.
(D) Seedling grown in continuous white light with a high red to far-red photon ratio. This spectral quality of light inhibits hypocotyl elongation and induces cotyledon expansion. This quality of light, which is often used in the laboratory, most closely resembles unobstructed sunlight outdoors.
(E) Seedling grown in continuous white light with a low red to far-red photon ratio. These light conditions mimic the shade environment, and the seedling undergoes a shade avoidance response, with an elongated hypocotyl and reduced cotyledon expansion.
(F) Seedling grown in continuous blue light. Blue light stimulates both the phytochromes and the blue light photoreceptors and is a very effective inhibitor of hypocotyl elongation and inducer of cotyledon expansion.

Phytochromes exist in two interconvertible forms, Pr and Pfr (Furuya, 1993; Vierstra, 1993; Quail et al., 1995). They are synthesized in the Pr form, whose absorption maximum is in the red (665 nm). Saturating red light converts 80% of the phytochrome to the Pfr form, which is the active form for most physiological responses and whose absorption maximum is in the far red (730 nm). By contrast, saturating far-red light leads to an equilibrium of ~97% Pr and ~3% Pfr. Under any light conditions except complete darkness, phytochromes are always present in an equilibrium of the two forms.

In Arabidosis, five distinct genes, designated *PHYA*, *PHYB*, *PHYC*, *PHYD*, and *PHYE*, encode the apoproteins (Clack et al., 1994; Quail et al., 1995). The expression patterns of the individual phytochromes are dramatically different at both the mRNA and protein levels (Quail et al., 1995). In particular, both *PHYA* mRNA and phyA protein accumulate to high levels in dark-grown seedlings, with exposure to light resulting in a >100-fold drop in phyA levels due to reduced gene expression as well as a higher turnover rate of the Pfr form of phyA than the Pr form. PhyA is thus referred to as a light-labile phytochrome. By contrast, both the expression and stability of other phytochrome species remain relatively constant in dark- and light-grown seedlings; thus, these are light-stable phytochromes.

Continuous red light alone can elicit photomorphogenic seedling development in wild-type seedlings, as shown in Figure 2B, although it is not as effective as some other qualities of light at inhibiting hypocotyl elongation. Phytochrome B appears to be the principal photoreceptor for continuous red light. Loss-of-function mutations at the *PHYB* locus cause a long hypocotyl phenotype in red light in Arabidopsis (*hy3* mutants; Nagatani et al., 1991a; Somers et al., 1991; Reed et al., 1993) and cucumber (*lh* mutants; Lopéz-Juez et al., 1992; Smith et al., 1992); conversely, *PHYB* overexpression causes a light-dependent short hypocotyl phenotype in Arabidopsis seedlings (Wagner et al., 1991; McCormac et al., 1993). These complementary results suggest that phyB mediates the inhibition of seedling hypocotyl elongation in response to red light. The phyB overexpression studies also indicate that the level of the photoreceptor is somewhat limiting in the plant, because the degree of hypocotyl shortening correlated with the increase in phyB level (Wagner et al., 1991).

Far-red light also effectively induces photomorphogenic seedling development, as shown in Figures 1A and 2C. The far-red high-irradiance response (FR-HIR) results in a short hypocotyl and open, expanded cotyledons. The cotyledons remain pale white, that is, without chlorophyll, because protochlorophyllide reductase requires short wavelength light energy for its catalytic action (Goodwin, 1988). It seems contradictory that far-red light, which converts most phytochrome to the presumably inactive Pr form, can promote photomorphogenesis,

(G) Seedling grown in continuous UV-A light. UV-A inhibits hypocotyl elongation and promotes cotyledon expansion. The cotyledons are darker than those of seedlings exposed to other wavelengths because UV-A light induces anthocyanin accumulation.

but a close look at the photobiological properties and expression pattern of phyA suggests that it is a suitable candidate (Quail et al., 1995). Under continuous far-red light, the low Pfr to Pr ratio (3% to 97%) actually helps to stabilize the size of the phyA pool because the Pr form of phyA is more stable than the Pfr form. This leads to a high overall level of phyA, and the 3% Pfr form from a large phyA pool could be sufficient to mediate physiological responses.

Indeed, experimental evidence strongly suggests that phyA is the principal, and possibly the only, photoreceptor involved in perceiving continuous far-red light and mediating the corresponding photomorphogenic responses. Mutants of the *PHYA* locus are essentially blind to continuous broad-band far-red light and exhibit a typical etiolated morphogenetic pattern similar to that of dark-grown seedlings (Nagatani et al., 1993; Parks and Quail, 1993; Whitelam et al., 1993). As with phyB, overexpression studies also provide complementary evidence for phyA's role in mediating the FR-HIR. Transgenic tobacco and Arabidopsis plants constitutively and ectopically overexpressing oat or rice *PHYA* display enhanced sensitivity to far-red light and a persistent FR-HIR in light-grown seedlings (Boylan and Quail, 1991; McCormac et al., 1991, 1992b, 1993; Whitelam et al., 1992).

Unlike *PHYB* overexpression, which increases seedling sensitivity to red light specifically, *PHYA* overexpression causes hypersensitivity to both far-red and white light (containing red light) in tobacco (Cherry et al., 1991; Nagatani et al., 1991b), tomato (Boylan and Quail, 1989), and Arabidopsis seedlings (Boylan and Quail, 1991). The white light–dependent short hypocotyl phenotype of the *PHYA* overexpressers appears to be due to the constitutive, ectopic expression of the apoprotein. It is unlikely that phyA normally plays a role in red light perception in wild-type light-grown plants, because *PHYA* expression levels are greatly diminished in deetiolated plants. Nevertheless, the *PHYA* overexpression results imply that a high level of phyA can nonspecifically activate the red light–specific phytochrome signaling pathway. Alternatively, it is possible that the different phytochromes share the same downstream signaling components but have distinct spatial locations and/or differ in their signaling effectiveness. As a result, ectopic expression of one phytochrome would mimic the endogenous function of another phytochrome.

The role of phyA in wild-type plants under natural environments is likely to be limited to the initial deetiolation of seedlings just emerging into the light from under a soil layer because the level of phyA decreases drastically with time. It is also possible that phyA is important for slowing down etiolated growth and initiating photomorphogenic growth when a seedling emerges from the soil into a light environment, such as deep shade, that is enriched in far-red light (see reviews in Quail, 1994; Quail et al., 1995; Smith 1995). Although phyB plays a dominant role in white light–grown plants, the remaining phyA seems also to have important, if limited, functions in processes such as day-length perception and control of gene expression (Johnson et al., 1994; Reed et al., 1994). The specific roles of phytochrome species other than phyA and phyB are still not clear.

Perception of Light Quality

The capability of higher plants to perceive changing light quality provides the basis for the shade avoidance response (Figures 1 and 2; Kendrick and Kronenberg, 1994). Because green plants absorb red light much more efficiently than they absorb far-red light, transmitted (shade) light and reflected light have lower ratios of red to far-red photons than does incident light (Figure 1; Smith et al., 1990). Most plants are very sensitive to changing light quality, and they can detect neighboring competitors by the quality of their reflected light up to a distance of at least 30 cm (Ballaré et al., 1987, 1990; Smith et al., 1990). Upon sensing low ratios of red to far-red photons, Arabidopsis seedlings respond with an acceleration of longitudinal growth (such as lengthening of the hypocotyl; Figures 2D and 2E) and a reduction in cotyledon and leaf expansion.

The major photoreceptor for detecting the ratio of red to far-red light appears to be phyB, possibly through changing the ratio of the Pr and Pfr forms. The best evidence for this hypothesis comes from the analysis of shade avoidance and end-of-day far-red responses in several *phyB* mutants, such as *hy3* in Arabidopsis (Nagatani et al., 1991a) and *lh* in cucumber (Lopéz-Juez et al., 1990; Whitelam and Smith, 1991; Smith et al., 1992). The end-of-day far-red response refers to the lengthening of the hypocotyl or stem caused by a pulse of far-red light to seedlings or plants at the end of each photoperiod. The phyB-deficient mutants exhibit reduced shade avoidance responses and reduced sensitivity to end-of-day far-red light treatments. However, because mutants defective in all phytochromes due to a chromophore biosynthesis defect are even more impaired in sensing the red to far-red photon ratio (Arabidopsis *hy1* and *hy2* mutants, Whitelam and Smith, 1991; tomato *aurea* mutants, McCormac et al., 1992a), phyB cannot be the only photoreceptor involved in light quality perception. Perhaps other light-stable phytochromes, such as phyC, phyD, and phyE, mediate the residual shade avoidance response in *phyB* mutants. PhyA does not seem to be involved in sensing light quality in wild-type light-grown plants, because *phyA* mutants appear to respond normally to low red to far-red photon ratios (Nagatani et al., 1993; Parks and Quail, 1993).

Early Phytochrome Signaling

Several genes have been identified genetically that may be involved in phytochrome signaling specifically (i.e., rather than signaling from all photoreceptors). The tomato *high pigment* (*hp*) mutant shows exaggerated phytochrome responses and is dwarfed and dark green (Peters et al., 1989, 1992). This phenotype is similar to that of transgenic tomato plants overexpressing phytochromes A and B (Boylan and Quail, 1989; Wagner et al., 1991). However, the *hp* mutant does not accumulate higher levels of phytochrome, nor is it defective in the degradation of phytochrome (Peters et al., 1992). The *hp* mutant displays a reduced threshold in the response to red light, suggesting that the *hp* mutation causes hypersensitivity to phytochrome stimulation (Adamse et al., 1989). These results

led to the hypothesis that the *HP* gene product is involved in an amplification step in phytochrome signaling and may act to inhibit responsiveness to phytochrome signaling. The increased sensitivity might be specific to a light-stable Pfr; it may also include light-labile Pfr forms (Peters et al., 1989).

Some of the participants in the transduction of signals from phyA specifically are beginning to be identified. Two loci, *FHY1* and *FHY3*, have recently been described that may be involved in transducing signals from phyA (Whitelam et al., 1993). *fhy1* and *fhy3* mutants are defective in continuous far-red-light–mediated inhibition of hypocotyl elongation, despite the fact that they have normal levels of functional phyA. Molecular characterization of these two loci will provide insight into the early events involved in phyA action and reveal the identity of components of the phyA-specific signal transduction pathway.

Evidence from microinjection experiments that tested a variety of known signaling molecules or their agonists and antagonists has indicated that trimeric G proteins, Ca^{2+}, calmodulin, and cGMP are possible components of the phyA signaling pathway (Neuhaus et al., 1993; Bowler and Chua, 1994; Bowler et al., 1994). These studies utilized the tomato *aurea* mutant, which appears to be deficient in all types of phytochrome (Parks et al., 1987; Quail, 1994; Whitelam and Harberd, 1994). *Aurea* mutant seedlings have a pale yellow-green color due to reduced chlorophyll content and a long hypocotyl when grown under normal light conditions, and they accumulate only negligible quantities of anthocyanins (Koornneef et al., 1985). In addition, the plastids in *aurea* hypocotyl cells fail to develop into normal chloroplasts (Kendrick and Nagatani, 1991), and the transcripts of phytochrome-regulated genes accumulate to only very low levels (Sharrock et al., 1988). Active phytochrome cannot be completely absent from *aurea* mutant plants, however, because they are able to undergo a qualitatively normal shade avoidance response (Kerckhoffs et al., 1992).

Remarkably, injection of purified oat phyA into the hypocotyl cells of *aurea* mutant seedlings results in the restoration of normal chloroplast development, photoregulated expression of a chlorophyl *a/b* binding protein (*cab*) reporter gene, and anthocyanin biosynthesis (Neuhaus et al., 1993). By injecting specific agonists or antagonists of well-defined signal transducers with or without oat phyA, it was possible to show that light signals perceived by injected phyA may result in the activation of one or more trimeric G proteins and that Ca^{2+}/calmodulin and cGMP appear to act downstream of phyA in both parallel and converging pathways to regulate anthocyanin biosynthesis, chloroplast development, and *cab* gene expression (Neuhaus et al., 1993; Bowler et al., 1994). Because Ca^{2+} and cGMP carry minimal signaling specificity on their own, it appears that hypocotyl cells are preprogrammed to respond photomorphogenically to these signaling intermediates.

It is important to keep in mind that, at least in light-grown Arabidopsis seedlings, phyA does not normally appear to be involved in controlling the developmental processes that oat phyA does in the tomato *aurea* mutant (Whitelam et al., 1993). However, as discussed earlier, when overexpressed in transgenic plants, phyA is capable of mediating responses normally attributed to phyB (Boylan and Quail, 1991); this might explain why oat phyA alone can rescue all aspects of the *aurea* mutant phenotype. Therefore, it will be of interest to determine whether these signal transducers are indeed restricted to phyA, or whether they were involved in signaling pathways for additional phytochrome species.

Blue Light Perception

Continuous blue light is effective in inducing photomorphogenic responses, as shown in Figure 2F. Although the blue light receptors are hypothesized to be flavin binding proteins, only very recently has one of them, the product of the *HY4* gene, been characterized at the molecular level (Ahmad and Cashmore, 1993). Mutations at the *HY4* locus cause a decrease in sensitivity to blue light, as evidenced by a dramatic long hypocotyl phenotype (Koornneef et al., 1980) and a marked decrease in cotyledon expansion (Blum et al., 1994; McNellis et al., 1994b) in response to blue light. The N-terminal half of the HY4 protein shows homology with bacterial photolyases, which are flavoproteins catalyzing blue light–dependent DNA repair reactions (Ahmad and Cashmore, 1993). This suggests that HY4 has the capacity to act as a blue light photoreceptor. The recent findings that insect cell–produced HY4 protein indeed associates with flavin adenine dinucleotide (Lin et al., 1995a) and that overexpression of the HY4 protein causes a short hypocotyl phenotype specifically in blue light (Lin et al., 1995b) further support the conclusion that HY4 is a blue light receptor mediating blue light inhibition of hypocotyl elongation.

It is almost certain that additional photoreceptors exist that mediate other blue light responses, because *hy4* mutants are not defective in any of those responses, including phototropism (Liscum et al., 1992; Liscum and Hangarter, 1994; Liscum and Briggs, 1995). Little, if anything, is known about immediate downstream signaling events for blue light responses, although it has been shown that a membrane-bound GTPase activity in pea seedlings can be activated specifically by blue light (Warpeha et al., 1991; Kaufman, 1993).

Ultraviolet Light Perception

UV light causes cotyledon expansion and dramatic hypocotyl shortening (Kendrick and Kronenberg, 1994). A schematic diagram of a seedling grown in continuous UV-A light is shown in Figure 2G. UV-B light is also very effective in reducing hypocotyl elongation, especially when provided as a supplement to white light (Lercari and Sodi, 1992). Plants also display numerous physiological responses that appear to involve the action of a specific UV-B photoreceptor (Mohr, 1994). These observations suggest that plants possess both UV-A and UV-B photoreceptors. However, analysis of responses to UV-A and UV-B light is complicated by the absorption of these wavelengths of light by both phytochrome and flavin-containing blue light receptors. In part because of these difficulties, the UV light receptors are the least understood of all the photoreceptors.

Studies of mutants defective both in phytochromes and in blue light perception have suggested that specific UV-A photoreceptors exist and that stimulation of UV-A photoreceptors inhibits hypocotyl elongation in Arabidopsis seedlings (Young et al., 1992). Similarly, analysis of responses to UV-B light in cucumber seedlings deficient in light-stable phytochromes suggests that a specific UV-B photoreceptor exists (Ballaré et al., 1991). It appears that in wild-type plants, phytochromes and UV light receptors work in conjunction to mediate responses to UV-A and UV-B light, and in a phytochrome mutant background, the ultraviolet photoreceptors assume primary importance (Ballaré et al., 1991; Lecari and Sodi, 1992). At present, it is not clear whether the UV-A and UV-B photoreceptors are separate entities or whether a single photoreceptor may be responsible for absorbing both UV-A and UV-B light. This situation would be clarified by the isolation of mutants specifically defective in UV-A or UV-B sensitivity.

DOWNSTREAM REGULATORS OF PHOTOMORPHOGENIC DEVELOPMENT

To achieve control of seedling developmental pattern, specific light signals perceived by photoreceptors must modulate the activities of regulatory molecules responsible for determining the developmental pattern of the plant at both the cellular and organismal levels. Molecular genetic studies, particularly with Arabidopsis, have identified a handful of these regulatory molecules. The cloning of genes involved in these downstream regulatory events has yielded several novel developmental regulatory proteins and has shed light on the mechanism of light modulation of plant developmental patterns. Although the information at present is fragmented, it seems to indicate that the complex array of light sensing and early signaling processes converges to common downstream regulators that in turn control cellular developmental decisions.

The Pleiotropic *COP/DET/FUS* Loci May Define Master Regulators That Repress Seedling Photomorphogenesis

If light signals were transduced to master developmental regulators that control the developmental switch between skotomorphogenic or photomorphogenic pathways, then mutations in those regulators should "lock" seedling development in one pathway independent of light. Screens for mutants exhibiting skotomorphogenic development in the light have yielded mainly photoreceptor mutations (see previous sections); in addition, most of the mutants recovered in these screens are only partially affected, retaining some aspects of photomorphogenic development in the light. Genetic screens for mutants that, conversely, exhibit photomorphogenic seedling development in the absence of light have yielded mutations at six loci. These dark-grown mutant seedlings exhibit the morphology and cell differentiation, plastid differentiation, and gene expression patterns of light-grown wild-type seedlings. These loci include *DEETIOLATED1* (*DET1*; Chory et al., 1989), *CONSTITUTIVE PHOTOMORPHOGENIC1* (*COP1*; Deng et al., 1991), *COP9* (Wei and Deng, 1992), and *COP8*, *COP10*, and *COP11* (Wei et al., 1994b). Interestingly, severe or null alleles of all of these loci also lead to high anthocyanin accumulation in the cotyledons of developing embryos and young seedlings, a classic characteristic of the *fusca* (*fus*) mutants (Müller et al., 1963). Indeed, it has recently been shown that each of the six pleiotropic *COP/DET* loci is identical to a previously identified *FUS* locus (Castle and Meinke, 1994; McNellis et al., 1994a; Miséra et al., 1994). In addition, mutants at four additional *FUS* loci also lead to pleiotropic constitutive photomorphogenic seedling development in darkness (Miséra et al., 1994; S.F. Kwok, B. Piekos, S. Miséra, and X.-W. Deng, unpublished results).

The recessive nature of the mutations at all 10 of the *COP/DET/FUS* loci suggests that they are required to repress photomorphogenic development in darkness and that light acts to abrogate their repressive function. The similar and pleiotropic nature of their mutant phenotypes implies that their products are required for related regulatory steps that control the primary switch from the skotomorphogenic to the photomorphogenic developmental pathway, that is, that they act before any major branch points of the regulatory cascades controlling specific aspects of light-regulated processes (such as cellular differentiation, plastid development, or hypocotyl elongation). Evidence from transgenic Arabidopsis lines moderately overexpressing *COP1* supports this hypothesis: these lines exhibit partial suppression of seedling photomorphogenic development under continuous far-red or blue light conditions (McNellis et al., 1994b). Because blue and far-red light effects are primarily mediated by HY4 and phyA, respectively, this result suggests that both of these photoreceptors can independently mediate light inactivation of the repressive activity of COP1. Therefore, these experiments provide direct evidence for the prediction that COP1 acts as a molecular repressor of photomorphogenic development (Deng, 1994) and that light signals perceived by multiple photoreceptors converge to mediate inactivation of COP1. Furthermore, genetic interaction studies with photoreceptor mutations have suggested that the pleiotropic *cop/det/fus* mutations are epistatic to mutations in phytochromes and the HY4 blue light photoreceptor (Chory, 1992; Wei and Deng, 1992; Ang and Deng, 1994; Wei et al., 1994b), suggesting that signals from multiple photoreceptors converge at or before these loci to inactivate their repressive action, as shown in Figure 3.

Two alternative hypotheses have been proposed to explain the possible relationships among the *COP/DET/FUS* loci, based on the fact that mutations in all of these loci result in almost identical seedling phenotypes. One possibility is that all of these proteins function in close proximity with each other in the same pathway. The synthetic lethality and specific epistatic interactions that have been observed between weak *det1* and *cop1* mutations are consistent with this hypothesis (Ang and Deng,

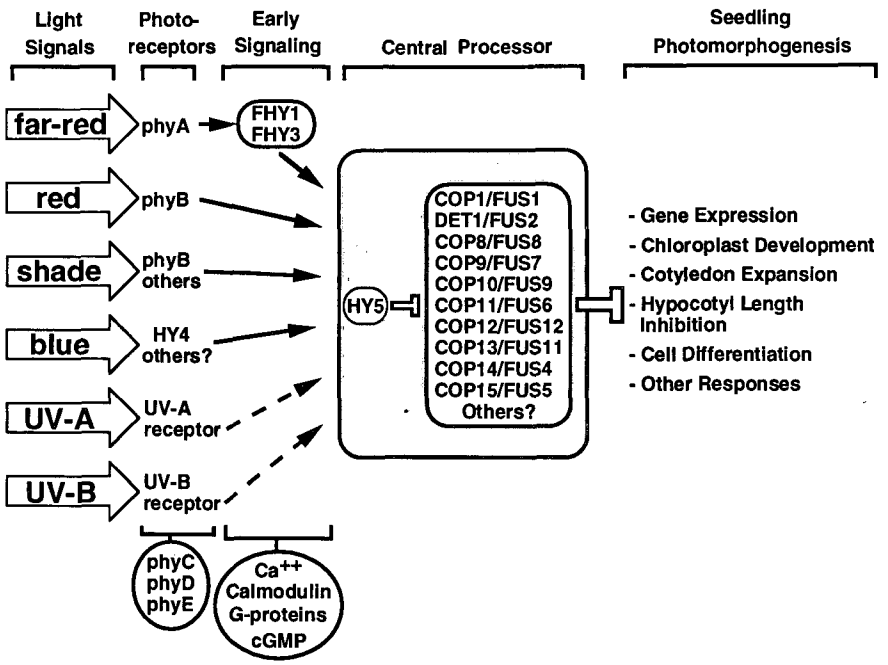

Figure 3. A Genetic Model of Light Control of Arabidopsis Seedling Development.

Light signals are perceived by an array of photoreceptors, some of which have been identified and others of which are unknown, that activate early signaling pathways. The specific roles of other phytochromes besides phyA and phyB are still not clear, nor is it known how many other receptors in addition to HY4 are involved in blue light perception. Although most of the early signaling pathways are represented by single arrows, they may involve multiple steps. G proteins, Ca^{2+}, calmodulin, and cGMP appear to be involved in several of these early signaling events, such as phytochrome signal transduction. The early signaling pathways converge at or before downstream components (COP/DET/FUS), which act as negative regulators of photomorphogenesis. Signals from the various photoreceptors relieve the repressive activity of the repressors, perhaps through HY5 and/or other signaling molecules. Note that UV-A and UV-B photoreceptors have not yet been isolated, but their existence is strongly suggested by genetic and physiological data. The possibility that these photoreceptors may also regulate the downstream repressors is hypothetical at this stage, as indicated by dotted arrows. It is also possible that a single photoreceptor absorbs both UV-A and UV-B light.

1994). Alternatively, these loci may define multiple parallel pathways that control the developmental switch from skotomorphogenesis to photomorphogenesis (Chory, 1993; Miséra et al., 1994). Those models are not necessarily mutually exclusive. It is possible that some of the genes act in one pathway, with others acting in different pathways.

One possible way for these gene products to function in the same pathway would be to act as a multisubunit protein complex. The molecular cloning of four pleiotropic *COP/DET/FUS* loci—*COP1* (Deng et al., 1992; McNellis et al., 1994a), *COP9* (Wei et al., 1994a), *COP11/FUS6* (Castle and Meinke, 1994), and *DET1* (Pepper et al., 1994)—has made it possible to test whether any of these gene products are found in a complex. In light-grown seedlings, the COP9 protein (22.5 kD) is a component of a large (~560 kD) light-stable protein complex (Wei et al., 1994a). In etiolated seedlings, some of the COP9 complex is shifted to a higher molecular mass. This higher molecular mass complex disappears within 5 min after irradiation with light. This finding raises the possibility that the COP9 complex in its higher molecular weight form represses photomorphogenesis in the dark and that light signals cause the partial dissociation of the complex, thereby relieving repression of photomorphogenesis. Interestingly, the COP9 complex is not detectable in extracts from *cop8* and *cop11* mutant seedlings, which suggests that the COP8 and COP11 proteins are necessary for the formation of the COP9 complex and perhaps even actual component proteins of the complex (Wei et al., 1994a).

Possible Mechanisms and Regulation of Pleiotropic *COP/DET/FUS* Gene Action

All four pleiotropic *COP/DET/FUS* genes cloned so far encode novel proteins, although they may have related counterparts in the animal kingdom (Chamovitz and Deng, 1995). COP1 possesses three well-characterized structural domains: a ring-finger zinc binding motif with the potential to bind to DNA, a coiled-coil domain with the potential to be involved in protein–protein interactions, and a domain with multiple WD-40 repeats characteristic of the β subunit of trimeric G proteins (Deng et al., 1992; von Arnim and Deng, 1993; McNellis et al., 1994a). The C-terminal half of COP1 bears significant homology with the $TAF_{II}80$ subunit of Drosophila TFIID, a component of RNA polymerase II (Dynlacht et al., 1993). The predicted

structural features of COP1 suggest that it may suppress photomorphogenic seedling development by directly regulating the transcription of genes involved.

Using the GUS reporter enzyme as a protein fusion tag, both DET1 and COP1 have been shown to be likely nuclear regulators (Pepper et al., 1994; von Arnim and Deng, 1994). Recently, the COP9 complex was also demonstrated to be nuclear through an immunolabeling assay using isolated Arabidopsis protoplasts (N. Wei and X.-W. Deng, unpublished data). Thus, it seems likely that all four cloned pleiotropic *COP/DET* genes encode nuclear regulators, raising the possibility that some or all of these proteins could control gene expression directly.

Studies using the GUS–COP1 fusion protein expressed transiently in the epidermal cells of onion bulbs or stably in hypocotyl cells of transgenic Arabidopsis seedlings suggest that the subcellular localization of COP1 may be regulated by light (von Arnim and Deng, 1994). The GUS–COP1 fusion protein accumulates in the nucleus in the dark and becomes depleted from the nucleus in the light. In Arabidopsis hypocotyl cells, the level of GUS–COP1 fusion protein in the nucleus changes in response to dark–light transitions and correlates quantitatively with the extent of repression of photomorphogenic development. In roots of transgenic Arabidopsis seedlings, the GUS–COP1 fusion protein is constitutively nuclear, consistent with the established role of COP1 in suppressing root chloroplast development in both the light and the dark (Deng and Quail, 1992). This observation supports a model in which COP1 acts in the nucleus to suppress photomorphogenic development, and it suggests that repression of photomorphogenesis by COP1 may be relieved by depletion of COP1 activity from the nucleus. It is possible that some of the other pleiotropic *COP/DET/FUS* genes are involved in mediating the light control of COP1 nuclear localization. This could be examined in *cop/det/fus* mutants expressing the GUS–COP1 fusion protein.

HY5, A Positive Regulator Acting Downstream of Multiple Photoreceptors

Mutations at the *HY5* locus cause a long hypocotyl phenotype in far-red, red, blue, and UV-A light, indicating that HY5 is required for mediating developmental responses to phytochromes and to blue and UV-A light receptors (Koornneef et al., 1980). This suggests that signals from phytochromes and the other photoreceptors converge at or before HY5 and that the role of HY5 is that of a positive regulator of responses to far-red, red, blue, and UV-A light. Genetic interactions between *hy5* mutations and severe or null pleiotropic *cop* mutations indicated that *HY5* probably acts upstream of *COP1*, *COP8*, *COP9*, *COP10*, and *COP11* (Ang and Deng, 1994; Wei et al., 1994a, 1994b). This raises the possibility that HY5 may be involved in the light control of COP1 nuclear localization and/or the activity or formation of the COP9 complex. Interestingly, double mutants between *hy5* and certain *cop1* mutations give allele-dependent interactions (Ang and Deng, 1994). For example, the *hy5* mutation can partially suppress weak *cop1-6* alleles, whereas severe (and possibly null) alleles of *cop1* suppress the *hy5* phenotype. These allele-specific interactions may indicate that the HY5 and COP1 proteins interact physically.

The Less Pleiotropic *COP/DET* Loci May Regulate Subsets of Seedling Photomorphogenesis

Mutations at three *COP* and two *DET* loci uncouple subsets of the photomorphogenic responses from light signals. Mutations at *COP2*, *COP3*, and *COP4* result in cotyledon expansion and development in darkness (Hou et al., 1993). However, these loci are not involved in plastid differentiation or in the regulation of hypocotyl elongation. The *cop4* mutation, but not the *cop2* and *cop3* mutations, leads to high-level expression of nuclear, but not plastid-encoded, light-inducible genes. The *cop4* mutant also has a defective gravitropic response, suggesting that light signaling and gravitropic signaling pathways may share some common elements. Mutations in the *DET2* locus cause plants to display a photomorphogenic morphology in darkness and result in the derepression of light-regulated gene expression but do not cause chloroplast development in the dark (Chory et al., 1991). Thus, mutations at the *DET2* locus demonstrate that chloroplast development is separable from other aspects of photomorphogenesis. The *DET3* locus seems to control morphological aspects of photomorphogenesis exclusively; *det3* mutants are unaffected in light-regulated gene expression and chloroplast development (Cabrera y Poch et al., 1993). It seems possible that these loci act downstream of the pleiotropic photomorphogenic regulatory loci and that they encode components in branched pathways regulating subsets of seedling morphogenic responses to light.

Possible Roles of the Pleiotropic *COP/DET/FUS* Loci Beyond the Suppression of Photomorphogenesis

Severe or null mutations at all of the pleiotropic *COP/DET/FUS* loci cause a *fusca* phenotype and lethality after the seedling stage, although certain alleles of some of these genes do allow the development of a small rosette of true leaves before senescence occurs. These loci are thus involved in other essential cellular processes besides the repression of photomorphogenesis in the dark (Castle and Meinke, 1994). Even weak *cop1* and *det1* mutant alleles result in dwarfed adults when mutant plants are grown under normal light conditions (Deng and Quail, 1992; Pepper et al., 1994). Several additional lines of evidence also indicate that the *COP/DET/FUS* genes play an important role in the growth of plants in the light. A study using somatic chimeras revealed that the COP1/FUS1 protein is necessary for normal cell expansion in subepidermal tissues and also for trichome formation (Miséra et al., 1994), suggesting that COP1 acts to modulate cell differentiation patterns and gene expression patterns in the light. Moreover, *fus* mutants show defective responses to other developmental stimuli in addition to light (Castle and Meinke, 1994). All of these

observations suggest a role for these genes beyond simply the suppression of photomorphogenesis. Therefore, the pleiotropic *COP/DET/FUS* gene products could be viewed as general developmental regulatory molecules whose activity is modulated by light. Alternatively, it is possible that other signal transduction pathways converge with the light signal transduction pathways to modulate the activity of the *COP/DET/FUS* gene products.

HYPOTHESES AND PERSPECTIVES

In recent years, some general themes have begun to emerge regarding the signaling network mediating light control of seedling morphogenesis. In this section, we summarize two such themes that are suggested by available experimental results. Our working hypotheses are based on studies dealing with the high-irradiance response (HIR) of seedlings, particularly with regard to hypocotyl and cotyledon morphogenesis, and therefore are intended to explain only those processes. Our models may not account for other light responses (Kendrick and Kronenberg, 1994), such as the low or very low fluence responses.

Photomorphogenic Seedling Development: The Default Pathway

Photomorphogenesis appears to be a default developmental pathway, which must be repressed in the dark to allow etiolation to occur (Wei et al., 1994a). This conclusion is supported by the isolation of recessive mutations at 10 pleiotropic loci that cause the plant to display nearly all aspects of photomorphogenic development in the absence of light. The *COP/DET/FUS* gene products are therefore postulated to act as general supressors of photomorphogenesis (Chory, 1993; Deng, 1994). In contrast, extensive genetic screens have never revealed any mutation that completely abolishes photomorphogenic seedling development, although this may be because such a mutant is likely to be lethal. *HY5* is the only locus in which mutations result in a decreased ability to deetiolate in response to red, far-red, and blue light. Taken together, these observations suggest that the master regulatory mechanism may be repressive in nature.

If photomorphogenic development is indeed the default pathway, and if photomorphogenesis must be repressed to allow skotomorphogenesis to occur, then environmental influences other than light might be expected to perturb the repressive machinery. In fact, a number of external stimuli other than light can cause photomorphogenic responses in darkness. Chory et al. (1994) reported that cytokinins enable dark-grown wild-type Arabidopsis seedlings to display some phenotypic features of *det1* mutants. Araki and Komeda (1993) found that constant shaking of liquid-cultured Arabidopsis seedlings in the dark can induce some photomorphogenic traits and eventually lead to flowering. In addition, cyclic heat treatment was reported to direct photomorphogenesis-like development in dark-grown pea (Kloppstech et al., 1991) and barley (Beator et al., 1992) seedlings. It is possible that in the absence of light signals, certain external stimuli, such as the presence of a phytohormone, cyclic heat treatment, or mechanical stimulation, may somehow reduce the activities of some of the suppressive components of photomorphogenesis and result in development according to the default photomorphogenic pathway.

This hypothesis is also consistent with the evolutionary history of green plants. Etiolation is a property of more highly evolved plants, such as angiosperms; more primitive plants usually are not able to etiolate and tend to follow similar developmental patterns in light and darkness. For example, gymnosperms and the great majority of algae form chloroplasts in the dark. Among those that do not, such as *Euglena* and *Ochromonas*, proplastid-like structures develop. These proplastid-like structures do not contain the extensive prolamellar bodies usually associated with etioplasts (Kirk and Tilney-Bassett, 1978). Skotomorphogenic development may therefore have evolved in response to terrestrial conditions such as soil and dense vegetation canopies. According to this scenario, photomorphogenesis is the original, default developmental pathway, whereas skotomorphogenesis is a specialized developmental pattern used to enhance adaptability to darkness and low light conditions.

The Quantitative Nature of the Light Regulatory Network

As summarized in Figure 3 and in previous sections, the pleiotropic *COP/DET/FUS* loci seem to regulate the primary switch between photomorphogenesis and skotomorphogenesis and act upstream of the branched pathways that regulate specific developmental processes, such as hypocotyl elongation, cotyledon expansion, and plastid differentiation. On the other hand, the COP/DET/FUS proteins act at or after the convergence of light signals perceived by multiple photoreceptors, including phyA, phyB, and HY4. The mechanism of this convergence of signals is largely unknown, but the activity of HY5, the activity of the COP9 complex, and the nuclear abundance or activity of COP1 are all potential targets for light modulation (Ang and Deng, 1994; McNellis et al., 1994b; von Arnim and Deng, 1994; Wei et al., 1994a). Thus, the pleiotropic *COP/DET/FUS* genes, *HY5*, and probably other as yet unidentified loci comprise a "nexus" region in the light regulatory network that serves to integrate light signals perceived by the various photoreceptors and control numerous developmental decisions.

One feature of the HIR is that the degree of the response generally correlates with the quantity of the light stimulus (Kendrick and Kronenberg, 1994). The available data hint at two possible bases for these quantitative responses. First, as in many other biological signal perception systems, the quantity of the signal (photons) can proportionally increase the total

number and/or percentage of the receptors in their activated form, thus relaying a quantitative output to the downstream components. Increased cellular concentrations of the photoreceptor would also increase the total number of photoreceptors in the activated form, because the equilibrium between active and inactive photoreceptors would be the same under continuous irradiation at a given light fluence rate. This is consistent with overexpression studies of the phytochromes (reviewed in Quail et al., 1995; Smith, 1995) and the HY4 blue light photoreceptor (Lin et al., 1995b), which demonstrated that increased photoreceptor concentrations cause hypersensitivity to light signals. Thus, the amount of photoreceptor can also modulate the degree of the response.

Quantitative activation of photoreptors may then result in the quantitative modulation of the repressor activity defined by the pleiotropic *COP/DET/FUS* genes. In this way, the repressors could dictate the extent of plant responses. The fact that mutations of different severity correlate with the degree of phenotypic defects in several loci is consistent with this hypothesis. In addition, the effects of *COP1* overexpression on Arabidopsis photomorphogenic development correlate well with COP1 protein levels (McNellis et al., 1994b), illustrating the feasibility of quantitatively modulating COP1 level (or activity) to achieve variable degrees of inhibition of photomorphogenic development. The modulation of COP1 activity could be accomplished through the regulation of the abundance of COP1 protein in the nucleus (von Arnim and Deng, 1994). Based on these results, it seems possible that the controlled inactivation of *COP/DET/FUS* gene products could provide one basis for the capability of plants to respond quantitatively to light signals.

Future Perspectives

It is immediately obvious from examining Figure 3 that our knowledge of the details of light signal transduction is sketchy, despite the dramatic increase in the pace of progress in this area. A number of key participants in the photoregulation of development have now been characterized at the molecular level, including the phytochromes, a blue light photoreceptor, and some of the molecules involved in transducing and integrating signals from the different photoreceptors and controlling developmental responses. However, many questions remain. For example, it will be of great interest to determine the mode of action of the photoreceptors and their immediate downstream components. Also, the nature of the convergence of signals from the different photoreceptors is completely unknown. Continued studies of signaling intermediates, especially those that are involved in transducing signals from multiple photoreceptors, such as HY5, may shed some light on this process. Finally, the signaling pathways linking the master COP/DET/FUS repressor molecules with the control of gene expression are currently the subject of intense study using light-regulated promoters and light-inducible transcription factors (Carabelli et al., 1993; Quaedvlieg et al., 1995). Although some of the components of plant light signal transduction pathways appear to be similar to components of signal transduction systems previously defined in animals or other organisms (Bowler et al., 1994), many others are novel in structure and probably in function (although some may also be shared between the animal and plant kingdoms; Chamovitz and Deng, 1995). The study of light signal transduction in plants may therefore provide new insights into the exciting world of signal transduction in biological systems in general.

Another issue that has rarely been mentioned but should be of great importance in understanding light control of seedling morphogenetic pattern is the specificity of cellular response. So far, most of the key players identified, including phytochromes, the HY4 blue light photoreceptor, and the products of the cloned pleiotropic *COP/DET/FUS* genes, seem to be present in most if not all cell types, although each cell type produces a distinct cellular response to a particular light stimulus. For example, under continuous light exposure, the hypocotyl cells of the seedlings cease elongating, guard cells differentiate, and cotyledon cells divide and differentiate (i.e., epidermal cells expand and stomatal structures mature). It is essential to learn what genes and genetic mechanisms underlie this cellular specificity in the light response.

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